

psychologically structured interviews, has been well documented. Measuring the serum prolactin level immediately following an attack has been advocated as a diagnostic aid and, when elevated, may prove useful in confirming epileptic seizures.

There is no widely accepted effective treatment for pseudoseizures. There has been a renewed interest in the use of hypnosis, but its effectiveness in this disorder must be carefully evaluated in a controlled fashion. Therefore, an accurate diagnosis remains the single most important step in managing pseudoseizures.

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REFERENCES

- Desai BT, Porter RJ, Penry JK: Psychogenic seizures. *Arch Neurol* 1982; 39:202-209
- Gummit RJ: Intensive neurodiagnostic monitoring: Role in the treatment of seizures. *Neurology* 1986 Oct; 36:1340-1346
- Ramani V, Gummit RJ: Management of hysterical seizures in epileptic patients. *Arch Neurol* 1982 Feb; 39:78-81
- Riley TL, Roy A (Eds): Pseudoseizures. Baltimore, Williams & Wilkins, 1982
- Trimble MR: Serum prolactin in epilepsy and hysteria. *Br Med J* 1978 Dec 16; 2:1682

Should Hypertension in a Patient With Acute Stroke Be Treated?

COMMON MEDICAL WISDOM, still widely taught and believed in this country, holds that elevated blood pressure should be lowered during ischemic stroke and certain other cerebrovascular disorders. Although this dogma has been questioned in recent years, most practicing physicians still use antihypertensive measures immediately after cerebral infarction and in patients with subarachnoid or intracerebral hemorrhage.

High blood pressure is very common early in the course of acute stroke, being found in 70% or more of these cases when seen initially. Of those patients not treated for hypertension, the great majority become normotensive within ten days. The blood pressure rise associated with the onset of cerebral ischemia apparently subsides as brain function recovers and autoregulation is restored. In such persons a sudden new blood pressure elevation may suggest a recurrence of brain ischemia. Bilateral carotid occlusion in animals is accompanied by an immediate rise in the blood pressure, which generally is proportional to the fall in cerebral perfusion. These observations indicate that hypertension in a patient with acute stroke often is a physiologic mechanism designed to protect ischemic brain by increasing the blood flow.

Cerebral autoregulation is impaired in blood vessels irrigating that part of the brain where the stroke has occurred. Thus, blood flow to this area becomes passively dependent on arterial pressure. If the blood pressure falls, perfusion of the ischemic area declines, thereby leading to a possible increase of the infarct and jeopardizing recovery. Decreased flow also may encourage further thrombus formation and could cause additional narrowing in the originally affected artery.

Many physicians believe that hypertension worsens the outcome of acute cerebral infarction, perhaps by causing forced vasodilation, bleeding and cerebral edema. There is little clinical evidence to support this thesis. On the contrary, data exist to indicate that hypotension during an acute stroke, either through medication or from other mechanisms, may worsen rather than improve the clinical outcome by extending the original infarct and impairing flow through the watershed area.

There is some uncertainty as to the specific indications for treating hypertension in patients with acute stroke. Some experts feel that blood pressures elevated to levels of 220/130 mm of mercury or more always should be treated. Most experts recommend that therapy for high blood pressure is essential in the face of myocardial insufficiency (especially congestive heart failure), severe renal damage, retinal bleeding and when hypertensive encephalopathy clearly is present. Hypertensive encephalopathy, a term coined by Oppenheimer and Fishberg in 1928 for recurrent seizures in patients with acute nephritis with elevated blood pressure, is uncommon, is probably due to a breakdown of cerebral autoregulation and is characterized by weakness, apathy, headaches, drowsiness, seizures, visual loss, transient and often fluctuating focal neurologic signs plus coma.

As regards subarachnoid hemorrhage, many authorities now believe that induced hypertension along with volume expansion is useful to counter vasospasm. There is no clear-cut evidence that reducing the blood pressure in patients with hypertensive intracerebral hemorrhage is or is not helpful, and some literature indicates that a high blood pressure should not be lowered in this situation.

The above discussion relates only to acute stroke. The fact that chronic hypertension is the most common and potent risk factor for cerebral infarction remains undisputed. There also is good evidence that treating hypertension reduces the occurrence of stroke, and there are strong indications in the literature that lowering blood pressure after a cerebral infarction reduces the risk for subsequent events.

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REFERENCES

- Lavin P: Management of hypertension in patients with acute stroke. *Arch Intern Med* 1986 Jan; 146:66-68
- Spence JD, Del Maestro RF: Hypertension in acute ischemic stroke: Treat. *Arch Neurol* 1985 Oct; 42:1000-1002
- Wallace J, Levy L: Blood pressure after stroke. *JAMA* 1981 Nov 13; 246:2177-2180
- Yatsu FM, Zivin J: Hypertension in acute ischemic stroke: Not to treat. *Arch Neurol* 1985 Oct; 42:999-1000

Panic Disorder

ONE OF THE more interesting and rapidly changing neuropsychiatric conditions involves a heterogeneous group of disorders currently subsumed under the general heading "Panic Disorder." Although defined in the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition (DSM III), as "agoraphobia, with or without panic attacks," many clinicians find this classification inadequate. It is anticipated that the revised edition of DSM III (DSM III-R) will be a more useful reference. This topic was reviewed in the March 1983 issue of this journal and interested readers are referred to it.

Symptoms have been described as being due to a "profound activation of the sympathetic nervous system." They are primarily cardiovascular and respiratory in nature: dyspnea, palpitations, chest pain or discomfort, choking or smothering sensations, or a more general reflection of autonomic nervous system dysfunction—that is, dizziness; feelings of unreality; paresthesias in hands or feet; hot and cold flashes; sweating; faintness; trembling or shaking; fear of dying, "going crazy" or doing something uncontrolled during an attack, or a sense of apprehension or impending doom. While usually lasting for only minutes, they may persist for hours. The symptoms are accompanied by varying degrees of